Days	Control			Quinapril			Hydralazine		
	LVMI	αM	βM	LVMI	αM	βM	LVMI	αM	βM
0	2.66	1.0	1.0	2.66	1.0	1.0	2.66	1.0	1.0
2	2.61	1.2	1.1	2.55	1.5	0.8	2.60	1.5	1.1
4	2.62	0.8	0.8	2.59	2.3*	0.5*	2.64	1.6	0.6
7	2.74	0.9	0.9	2.37*	1.9*	0.7	2.48	2.1*	0.6
10	2.74	0.9	1.3	2.39*	2.5*	0.5*	2.81	1.7*	0.5*
21	2.70	1.2	1.2	2.35*	2.5*	0.5*	2.58	1.9*	0.4*

LVMI-LV mass index. *$p < 0.05$, compared with day 0 (same group) or control

These data show that reduction in hemodynamic load induced a change in myosin isoforms to adult pattern whereas only Q reduced LVMI, suggesting that LVH reversal may not be dependent solely on hemodynamic load.

933-99 Chronic Hydralazine or Enalapril Normalize Myosin Isoforms in the Spontaneously Hypertensive Rat, but Only Enalapril Normalizes Left Ventricular Mass

Garrie J. Haas, Margaret Ginn Pease, Melinda Hunnicutt, Mary E. Alton, A. John Merola, Robert J. Cody. *The Ohio State University, Columbus, Ohio*

Increased V3 myosin isoform is seen in left ventricular (LV) hypertrophy (H), but reversion to V1 with chronic afterload reduction and LVH regression is not well defined. We randomized SHR at 3–4 months to oral hydralazine (25 mg/kg), enalapril (40 mg/kg), or placebo. Age matched Wistar Kyoto rats were controls. Group average systolic blood pressure (mmHg) at baseline was 207 (Plac), 223 (Hydrl), 225 (Enal), and 156 (Cntrl). Rats were studied after 3 months of treatment. Echocardiographic LVmass (LVM) was from the cubic function: $LVM\ (mg) = 1.04[(EDD + PWT + IVS)^3 - EDD^3]$, corrected for body weight (LVMI), since echocardiography permits better identification of differing patterns of hypertrophy. Terminal hemodynamics were volumetric aortic flow (mean: MAF, ml/min) and Millar aortic pressure (mean: MAP). Systemic arterial resistance (SAR) and LV contractility (+dp/dt) was derived from digitized waveforms. Following termination, LV was harvested and frozen at −800° C, and myosin isoforms were determined by gel electrophoresis, with expression of V1, V2, V3 isoforms as % total. Analysis of variance was performed and group mean values are given below:

Group	N	MAP	MAF	SAR	+DP/DT	LVMI	V1	V2	V3
Placebo	17	151*	105*	1.57*	16246*	2.98 ns	32*	23	45*
Hydralazine	18	104	164	0.67	8562	2.80	68	15	17
Enalapril	13	94	144	0.68	8633	2.19#	80	11	9
Control	11	85	179	0.49	13022	2.55	60	15	25

*$p < .05$, placebo vs Hydrl, and vs Enal, #$p < 0.05$ placebo vs Enal

Thus, myosin isoform expression reverted to an adult pattern, in response to long term afterload reduction. Changes in myosin transcriptional patterns however, require additional assessment. Eccentric hypertrophy accounted for persistent LVMI following hydralazine, whereas greater LVMI reduction occurred with ACE inhibition. This suggests that factors other than afterload reduction contribute to LVMI regression with ACE inhibition.

933-100 Effect of 6 Antihypertensive Drugs on ECG Left Ventricular Hypertrophy

Vasilios Papademetriou, Elaine Der, John Gottdiener, Barry Materson, Domenic Reda, Barry M. Massie, VA Monotherapy of Hypertension Study Group. *VAMC and UCSF, San Francisco, CA; Georgetown University Medical Center, Washington DC*

Both ECG and echo LVH are markers of increased risk, but there is much less information on interdrug differences in ECG LVH regression. Therefore, changes in ECG LVH prevalence were examined after 3 mos titration phase and a 1 yr maintenance phase of randomized double-blind treatment with atenolol (AT), captopril (CP), clonidine (CL), diltiazem (DT), hydrochlorothiazide (HZ), prazosin (PZ) or placebo (PL) in 570 men with mild-moderate hypertension. The prevalence of Sokolow-Lyon (SL) and Minnesota code (MC) criteria were evaluated, as well as precordial voltage ($S_{V1} + R_{V5}$). Percent changes from baseline were:

	% of patients with LVH						Precordial Voltage (mV*10)		
	SL+			MC					
RX	Base	3 mos	1 yr	Base	3 mos	1 yr	Base	3 mos	1 yr
AT	12	+9*	+9	17	+7#	+15	27.1	+0.6	−0.1
CP	23	−8*	−4	33	−7	−4	28.1	0	+0.2
CL	16	+3	0	22	+5	+2	28.4	+0.3	−0.4
DT	18	+5	+8	29	+6	+16@	30.0	−0.9	−0.5
HZ	26	−5	−9	41	−8#	−9@	29.4	−0.5	−1.5
PZ	23	+1	−4	33	−1	+3	28.4	−0.1	+0.7
PL	16	+5	–	25	+7	–	28.3	+0.4	+1.1

* AT v CP 3 mos, # HZ v AT 3 mos, @ HZ v DZ 1 yr ($p < 0.05$)

Thus, HZ caused a decrease in LVH prevalence, while AT and DT caused an increase. These changes are concordant with echo observations, where HZ was the only drug to significantly reduce LV mass for equal levels of systolic BP reduction.

720 Complications of Coronary Intervention: Prediction and Management

Monday, March 20, 1995, 4:00 p.m.–5:30 p.m.
Ernest N. Morial Convention Center, Room 90

04:00

720-1 Incidence of and Factors Associated with Abrupt Closure in Patients Undergoing Elective, New Device Angioplasty in Native Coronary Arteries

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To determine the incidence and predictors of abrupt closure (TIMI flow = 0 or 1) in patients undergoing new device angioplasty (NDA), we studied the hospital records and qualitative morphologic and quantitative angiographic methods of 1,983 patients with unfavorable lesion morphologies treated electively in native coronary arteries with intracoronary stents (N = 96), extraction atherectomy (TEC; N = 27), directional atherectomy (N = 563), rotational atherectomy (N = 775), or laser (N = 522). The overall incidence of abrupt closure was 3.9% (78/1983), with 2.1% (42/78) occurring during the procedure and 1.8% (36/78) occurring out-of-lab (1 patient with both in- and out-of-lab abrupt closure). Among NDA, TEC (14.8%) and stents (8.3%) had relatively higher frequencies of abrupt closure compared to other NDAs (mean of 3.5%). Multivariate analysis of demographic and lesion characteristics revealed the following predictors associated with either in-lab or out-of-lab abrupt closure:

Independent Predictors	Odds Ratio	95% C.I.	p value
Unstable angina	2.01	1.13–3.57	<0.05
Lesion length ≥10 mm	2.00	1.26–3.19	<0.005
Angulation ≥45°	1.82	1.09–3.04	<0.05

C.I. = confidence interval

The sole independent predictor for out-of-lab only abrupt closure was dissection (Odds Ratio = 3.11, $p < 0.005$). *We conclude that:* (1) The incidence of abrupt closure (both in- and out-of-lab) was 3.9% in patients undergoing elective new device angioplasty, with nearly equal distribution of in- and out-of-lab occurrences: (2) Unstable angina, lesion length ≥10 mm, and angulation ≥45° predicted either in- or out-of-lab abrupt closure, whereas dissection was the sole significant predictor of out-of-lab only abrupt closure: and (3) both lesion-related (thrombus-containing lesions for extraction atherectomy) and device-related factors (stents with subacute thrombosis) added to the acceptable rates of abrupt closure with these new devices for lesions unfavorable for conventional angioplasty.

04:15

720-2 Predictors of Acute Complications After Percutaneous Coronary Revascularization with New Devices

Peter A. McCullough, William W. O'Neill, Melissa May, Anne Lichtenberg, Michelle Strzelecki, Cindy L. Grines, Robert D. Safian. *William Beaumont Hospital, Royal Oak, MI*

Studies of conventional balloon angioplasty (PTCA) have identified female gender, age, unstable angina, prior coronary bypass surgery, diabetes, lesion thrombus, and dissection as predictors of acute complications, but predictors of the risks of new device interventions are unknown. New devices